AMENDMENTS TO THE SPECIFICATION

Please replace the first paragraph beginning on page 7, ll. 1-5, with the following rewritten paragraph:

It is known that a very small proportion of donor T-cells possess the capability to recognize host alloantigen ~~(estimated to be less than 0.0%)~~. The present invention seeks to eliminate this response (render such cells non-responsive or tolerized to alloantigen or xenoantigen) by functionally altering the population of T-cells with allo- or xenoantigen reactive capabilities.

Please replace the second full paragraph beginning on page 11, ll. 12-17, with the following rewritten paragraph:

Supernatants from vogue cultured cells from the experiment shown in Figure 1 were analyzed for the concentration of interleukin 2 (IL-2). These results are contained in Figure 1A. Supernatants were analyzed by ELISA (R&D Systems, Minneapolis, MN). Supernatant concentration in pg per ml were shown on the y axis and the days of MLR culture on the x axis. The ~~additional~~ addition of anti-gp39 mAb inhibited IL-2 production from donor T-cells in primary MLR culture.

Please replace the second full paragraph beginning on page 12, ll. 12-23, with the following rewritten paragraph:

At the end of the primary MLR culture, cells were washed and replated at a concentration of 3×10^4 per 96 well plate. To each well, irradiated splenocytes from C57BL6 mice were added at a concentration of 10^5 cells per well. These results are contained in Figure 3A. Where indicated, IL-2 is added at a final concentration of 50 units per ml. The media consisted of 10% fetal calf serum, 5% supplements, and 2-ME. Microtiter wells were labeled with one microcurie per well at the indicated times for a period of eighteen hours prior to harvesting. On the y-axis are the mean proliferation values (Δ CPM) and on the x-axis are the days of secondary MLR culture. As can be seen from the results in ~~Figure 3H~~ Figure 3A, donor T-cells exposed to anti-gp39 mAb in primary

but not secondary culture retained alloantigen specific hyperresponsiveness in the secondary culture. This was reversible by the addition of exogenous IL-2 in the secondary culture alone.